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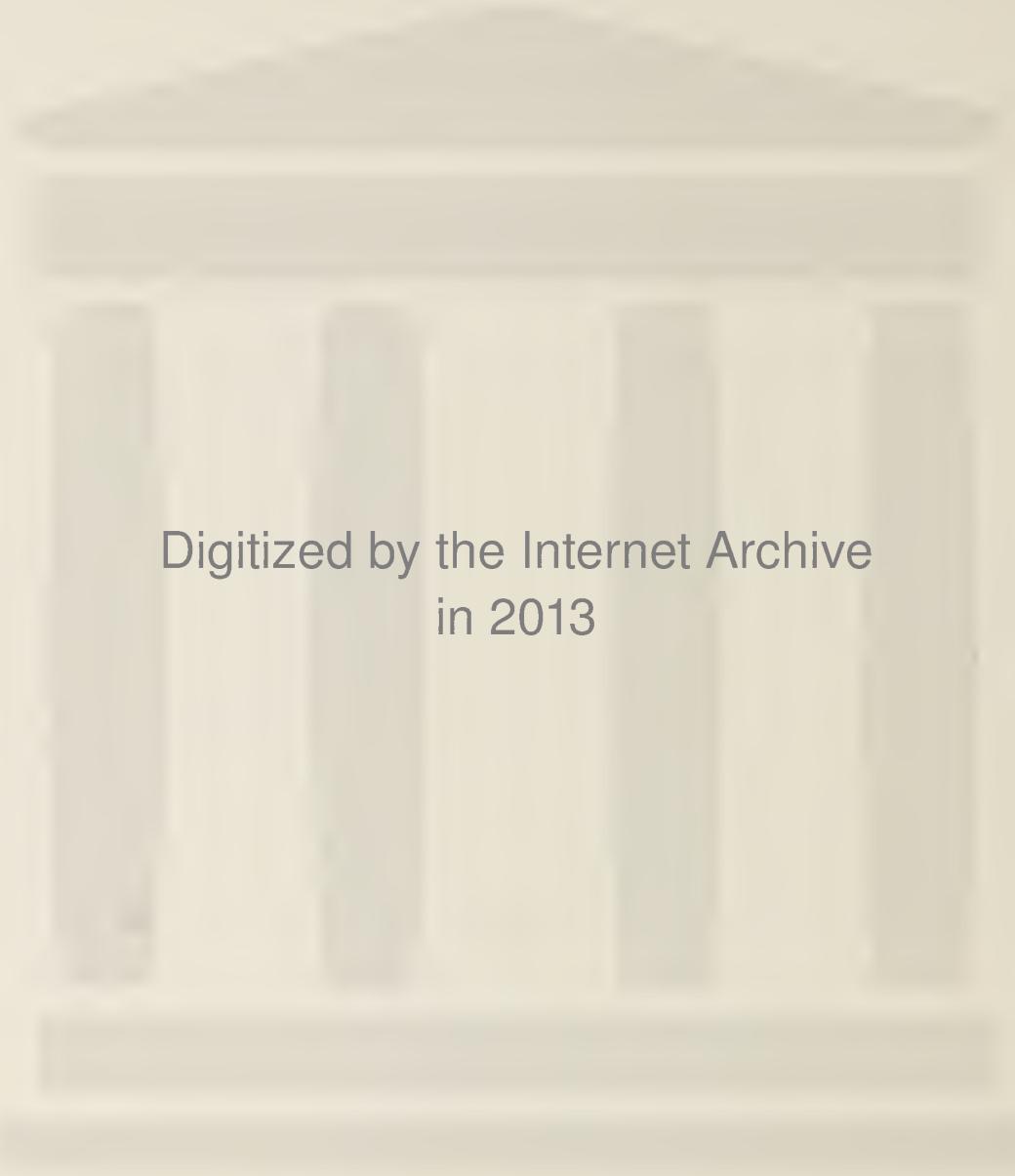
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Stock-poisoning plants of western Canada



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Stock-poisoning plants of western Canada

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The dots on the map represent
Agriculture Canada research
establishments.

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SUMMARY

This bulletin describes the most common plants that cause poisoning and injury in livestock as well as several plants that are potentially dangerous. Color photographs of the most common poisonous plants are provided. Line drawings illustrate less common plants. Recommendations aimed at prevention of stock poisoning are given. Brief descriptions of the toxic principles are provided and possible treatments are suggested.

RÉSUMÉ

Le présent bulletin décrit les plantes les plus communes qui provoquent l'intoxication et d'autres accidents chez le bétail ainsi que plusieurs plantes qui pourraient en menacer la santé. Les plantes vénéneuses les plus communes sont illustrées par des photos couleur et les plantes moins communes, par des dessins. On y présente des recommandations de nature préventive, on décrit brièvement les substances toxiques en cause et on propose des traitements possibles.

INTRODUCTION

Poisonous plants contain or produce poisonous substances that harm livestock. Depending on the poison and the amount of plant material eaten, an animal may die, may be disabled permanently, or may recover completely. Some plants cause immediate sickness or death after they have been eaten; with others poisoning symptoms may not become apparent for several days. A few plants contain poisons that are excreted almost immediately; these cause sickness or death only when large amounts are eaten in a short time or under certain other conditions. Still other plants can cause wounds or skin eruptions.

Poisonous plants are found in every ranching district. Fortunately, animals seldom eat poisonous plants except on overgrazed ranges or under unusual circumstances.

Another possible source of stock poisoning is the drinking water available to stock. When water is high in nutrients, either naturally or as a result of pollution, algae multiply rapidly; and when blue green algae are abundant, poisonous substances are excreted into the water.

Poisoned animals are usually hard to treat. There is often no known antidote or none that can be given under range conditions. Usually the first evidence of poisoning is discovery of the dead animal, often in remote areas. The best way to prevent losses is to know the common poisonous plants. The most important poisonous plants are described in this publication.

Consult a veterinarian when stock die. He can determine the cause of death and you will be able to prevent further losses. When you suspect that a plant is poisonous, take or send specimens of it to your district agriculturist or nearest research station. Positive identification can be made and control measures suggested. In cases of fatal poisoning, it is often possible to determine the cause from samples of the rumen content.

Pasture management in relation to poisonous plants

There are many poisonous and potentially poisonous plants but, with the exception of arrow-grasses, larkspurs, spotted and western water-hemlocks, and death camas, few regularly cause death.

Heavy losses caused by poisonous plants are usually associated with one of the following conditions.

OVERGRAZING Overgrazing causes good forage species to diminish in number and in size and less palatable plants to increase. Therefore, to control poisonous plants and to reduce livestock losses, maintain a productive pasture. Overgrazing not only occurs from having too many animals on too small a pasture but can also be the result of untimely grazing.

Pastures that are grazed too early in the spring, before the forage species have made much growth, soon become overgrazed. When native pastures

are grazed before they are ready for use, the earliest-growing species are forced to carry the grazing load. Death camas, low larkspur, saskatoon, choke cherry, and greasewood are some of the earliest range plants to show green leaves. On the prairies, livestock losses from plant poisoning in early spring are usually caused by these species. In April and early May losses can often be traced to pasturing too early over ground infested with death camas. In sandhill areas many lambs have been lost as a result of browsing on choke cherry or saskatoon. Losses ceased when flocks were moved to pastures where these species were not present.

MOVEMENT OF HUNGRY LIVESTOCK Movement of livestock at any season of the year, and particularly during the spring and early summer, gives the animals an opportunity to graze poisonous plants, and areas known to be infested with poisonous plants should be avoided. When animals are moved, their grazing habits are disturbed. If they are hungry, they are apt to eat large amounts of easily available forage and to graze plants that they refuse in their home pastures. In pastures where seaside arrow-grass occurs around drinking places, heavy losses can occur when cattle are herded into the pasture hungry and thirsty. Providing water and salt near the entrance gate can prevent these losses.

DROUGHT Losses from poisonous plants may increase during drought because lack of feed forces stock to eat any plant that is available. Where soils are high in selenium, poisoning through plants that accumulate this element can occur, especially after drought. Because selenium-containing plants remain poisonous in hay, livestock producers should check for the presence of these plants if it becomes necessary to feed poorer-quality hay.

In drought years plants such as arrow-grasses and low larkspur may be sparse, and poisoning becomes rare. However, the presence of these plants constitutes a danger at all times, and good management can prevent stock losses when return of good moisture conditions results in rapid increases in abundance of poisonous plants.

Control of poisonous plants

Control of poisonous plants by methods other than pasture management is not practiced widely. Grubbing is a practical way to eradicate water-hemlock and tall larkspur, and it is used on some ranches and in Prairie Farm Rehabilitation Act community pastures. However, all other species have such a wide distribution that grubbing is impractical. Where death camas and low larkspur are troublesome, eradication by cultivation followed by reseeding of grasses or grass-legume mixtures is suggested when soil and climate conditions are suitable. Although the cultivation of extensive range areas for summer pasture may not be warranted, cultivation and reseeding of selected areas for spring pasture is economical. This practice increases pasture production and provides range that is free from poisonous plants.

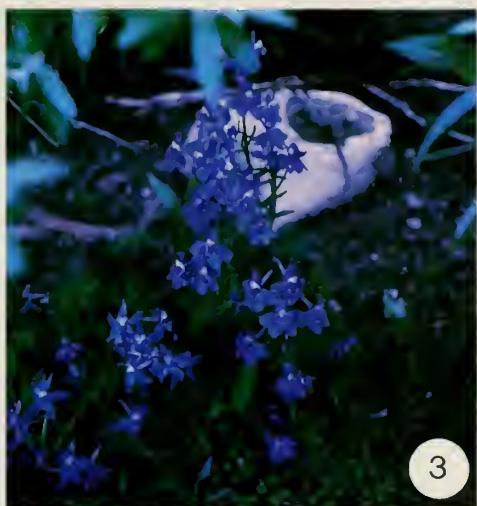
In Western Canada only a small amount of research has been attempted on the use of herbicides for the control of poisonous plants. Because the native sward is a complex mixture of forages and weeds, and because many



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Fig. 1 Seaside arrow-grass.

Fig. 2 Death camas.

Fig. 3 Low larkspur.

Fig. 4 Timber milk-vetch.



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Fig. 5 Spotted water-hemlock.

Fig. 6 Early yellow locoweed.

Fig. 7 Choke cherry.

Fig. 8 Saskatoon.

herbs and shrubs are valuable forage plants, both for livestock and wildlife, the loss of feed is often greater than the gain.

Applications of herbicides heavy enough to kill pasture weeds affect the seed setting of native grasses and reduce the numbers of certain palatable broad-leaved plants.

MAJOR TROUBLEMAKERS

Seaside arrow-grass (Triglochin maritima L.)

Fig. 1

DESCRIPTION Seaside arrow-grass is an erect, rushlike or grasslike perennial marsh herb 15-80 cm tall. It is clumped and unbranched. The leaves are basal, shorter than the flower stalks, and grow from a base covered with sheaths of old leaves. The new leaves are rushlike, thick, flat on one side and rounded on the other, spongy inside, smooth, shiny, and bright green. The flowers are small and greenish, clustered along spikelike flower stalks. The seed pods are three-sided, about 6 mm long, lobed, and oblong.

DISTRIBUTION AND HABITAT Seaside arrow-grass is common in salt marshes and alkaline sloughs throughout Western Canada. It grows with sedges, rushes, and slough grasses.

CONDITIONS OF POISONING This plant starts to grow earlier in the spring than do the associated grasses. It also has a more rapid regrowth than do these grasses after it has been mowed. Therefore, it is more dangerous at these times than at other growth stages, although stock craving salt may eat it readily at other times for the high amount it contains. Seaside arrow-grass also becomes especially dangerous late in the season after a sharp frost or in times of drought, generally whenever the plant is placed under stress. All parts of the plant are toxic, and young flowering stalks appear to be most palatable. In hay, the plant gradually loses its toxicity. Cattle are the livestock most often adversely affected by this plant, but sheep and horses are also susceptible to it.

TOXIC PRINCIPLE Seaside arrow-grass is one of a group of cyanogenic plants. These plants form glycosides which, through the action of enzymes, produce prussic acid (hydrogen cyanide, HCN). Under normal conditions, the glycoside and enzyme occur separately in the plant tissues, but under conditions of stress, breakdown of plant cells allows the substances to mix. The glycoside in Triglochin has been shown to be triglochinin, rather than amygdalin, as reported earlier. Amygdalin is common in several species of the rose family. The amount of HCN that can be formed differs in the various parts of the plant and depends on several factors, including growth stage, alkalinity of the soil, and moisture conditions.

SYMPTOMS AND LETHAL DOSE Symptoms of cyanide poisoning include rapid or deep breathing, muscular spasms, staggering, convulsions, and coma. Death follows from asphyxia or, when the dosage is large, from respiratory paralysis. The lethal dose of HCN is 0.2-0.35 g/100 kg body weight. Because fresh plants

can contain as much as 0.07% potential HCN, 3-5 kg of fresh plants can be enough to kill a 500-kg animal. The lethal dose for sheep is about 1-1.5 kg fresh plant material per 50 kg body weight.

TREATMENT Poisoning usually occurs too rapidly for treatment. Good results, however, have been obtained in cattle and sheep by intraperitoneal injections of sodium nitrite and sodium thiosulfate.

RELATED SPECIES Another species, marsh arrow-grass, Triglochin palustris L., is sometimes found in marshy places throughout Western Canada but is not nearly so common. It has been found in abundance in the lower Cariboo district in British Columbia, and losses have been attributed to it there.

Death camas (Zygadenus gramineus Rydb.)

Fig. 2

DESCRIPTION Death camas is a member of the lily family. It is a small, slender perennial herb 20-50 cm tall, with smooth, basal, yellowish green, grasslike leaves 3-8 mm wide, V-shaped in cross section, and thicker than those of grasses. The many creamy yellow flowers are about 6-8 mm long, lilylike, and borne on short stalks along the main spikelike stem. The plant grows from a dark-coated bulb, which resembles an onion and is 2-4 cm long. The bulb is generally 10-15 cm below ground. The plant reaches grazing height early in the spring, before most of the grasses. It flowers in May and early June. Seeds are formed in three-lobed capsules about 7-12 mm long during the latter part of June and early July, after which the plants quickly die back and disappear. Death camas may be confused with the wild onion, which, however, can be distinguished by its pink flowers, round head, and onion odor.

DISTRIBUTION AND HABITAT Death camas is common in Saskatchewan and Alberta. The usual habitat is in upland draws and depressions. Death camas is generally scattered over the range. As it seldom occurs to the exclusion of other species, it is hard to control.

CONDITION OF POISONING Because the plants reach grazable height before most grasses, they are most dangerous during the spring. All parts of the plant are poisonous, especially the bulbs. The plant is particularly dangerous when the soil is wet, because the bulbs may be easily pulled up then. Cattle are occasionally poisoned early in the spring before green grass is available and when the soil is wet, but sheep are the animals that are most often poisoned.

TOXIC PRINCIPLE The poisonous principle is a group of steroid alkaloids, including zygacine and zygadenine.

SYMPTOMS AND LETHAL DOSE The symptoms that indicate poisoning by death camas are increased rate of breathing, excessive salivation, and nausea, often followed by vomiting, and afterwards by staggering and finally prostration. Animals may remain in a coma for hours or even days before death. Body temperature increases during initial stages but it drops to below normal upon onset of the coma and remains there until death occurs. About 0.3 kg of fresh plants per 50 kg body weight constitutes a lethal dose for sheep. The seeds

are especially poisonous; 50 g of seed is enough to cause the death of a mature cow. The plant is also toxic in the dried state and is poisonous in hay.

TREATMENT Subcutaneous administration of atropine sulfate and pictotoxin is recommended early in the poisoning stage. Most important, however, is learning how to recognize the plant and to keep animals away from it.

RELATED SPECIES Two other species of camas occur in the area but they are not widespread. The conditions of poisoning and the effects on stock are similar to those of death camas.

Meadow death camas, Zygadenus venenosus S. Wats.. is similar to death camas. This species occurs in the range area of British Columbia at elevations up to 1200 m and is found principally on moist grassy hillsides and in draws in the fescue grassland zone in fairly rich, well-drained soils.

White camas, Zygadenus elegans Pursh, resembles death camas in appearance but is considerably taller, 60 cm, and more robust. The leaves are 6-12 mm broad and bluish green. The flowers are larger than those of death camas, 10-15 mm across, greenish or whitish cream, and are more scattered along the main stem, often occurring on branched stalks. White camas is found throughout Western Canada but it is not common. It prefers moist locations in rich soils.

Tall larkspur (Delphinium glaucum S. Wats.)

Fig. 9

DESCRIPTION Tall larkspur, a member of the buttercup family, is a tall, stately perennial forb with stems growing to 2 m tall. The leaves are toothed, deeply cleft into three to five main lobes, and are alternate on the stem, with short hairs on one or both sides; the lower leaves are 8-20 cm in diameter and round in outline. The flowers are pale blue to purplish and have a characteristic spur. The fruit is composed of three upright compartments, each opening lengthwise to allow the many seeds to escape. The roots are long, fibrous, and woody. The plant starts to grow shortly after the snow melts.

Tall larkspur is often confused with monk's-hood and sticky purple geranium. The flowers of monk's-hood are hooded and those of sticky purple geranium are pink and do not occur on long stalks.

DISTRIBUTION AND HABITAT Tall larkspur has not been reported from Manitoba or southern Saskatchewan, but it is common in the western boreal forest, in the foothills of the Rocky Mountains, and in the central interior of British Columbia. It is less common or rare in the boreal forest of eastern Alberta and western Saskatchewan. It occurs in the margins of aspen-poplar groves, in open forest of balsam poplar, spruce, or pine, and in willow thickets, as well as in grassy openings in the forested areas. It requires some shade, fairly rich soil, and good moisture conditions.

CONDITIONS OF POISONING Tall larkspur is among the first plants to emerge in spring, and its foliage is highly palatable to livestock. Hence, cattle losses are heaviest early in the summer, when tall larkspur forms a large part of the



Fig. 9 Tall larkspur

available forage. Horses occasionally eat tall larkspur but usually do not ingest a lethal dose. Sheep can graze tall larkspur without evident damage; because of this apparent immunity, sheep have been widely used in the control of larkspur.

TOXIC PRINCIPLE Tall larkspur contains several diterpenoid alkaloids. One of these, delphinine, was long held to be the toxic principle, but more recent studies have shown that the responsible substance is methyllycaconitine.

SYMPTOMS AND LETHAL DOSE The symptoms of poisoning are nausea, general weakness, muscular twitching, and prostration. Poisoned animals when prostrated make desperate attempts to regain their feet. Fatal poisoning terminates in death, either from respiratory paralysis or from asphyxiation. The lethal dose is low: only about 2 kg of fresh young plants for a yearling, or about 3.5 kg for a mature cow. Mature plants are considerably less poisonous and also much less palatable. The seeds contain a very high concentration of poison but are not likely to be consumed in toxic quantities.

TREATMENT Medicinal treatment can be used, but, to be effective, it should be started very soon after poisoning has occurred. An affected animal should be kept quiet with its head up hill. Subcutaneous injections of physostigmin salicylate, pilocarpin hydrochloride, or strychnine sulfate are beneficial if the animal is not unduly excited by treatment.

Low larkspur (Delphinium bicolor Nutt.)

Fig. 3

DESCRIPTION Low larkspur is a member of the buttercup family. It is a beautiful, somewhat pubescent native perennial forb, with stems growing up to 50 cm high. The leaves are mostly basal, alternate, deeply divided into a number of linear segments; they grow up to 5 cm in diameter and are round in outline. The flowers are up to 3 cm across, blue to purple, with a long (15-20 mm) spur at the bottom. There are few to 15 flowers on short stalks along the main flowering stem. The fruit is similar to that of tall larkspur but is covered with short, fine hairs. The plant starts growth in early spring, generally before most of the grasses. Blossoms appear in May and early June, and seeds are formed in the latter part of June or early July, after which the plants quickly die back and disappear. Another low larkspur, Delphinium nuttallianum G. Pritz., occurs in the interior of British Columbia.

DISTRIBUTION AND HABITAT Low larkspur has not been reported from Manitoba or eastern Saskatchewan. It is common in the Wood Mountain area, west to the Cypress Hills and foothills of the Rocky Mountains in southern Alberta. In British Columbia it is widespread in the fescue grasslands of the east Kootenay and is replaced by D. nuttallianum farther west. Both species occur generally at altitudes of about 700-1500 m; they are found in mesic grasslands, between shrubbery, and on moist coulee slopes with rather rich, deep soils.

CONDITIONS OF POISONING Low larkspurs are among the earliest-appearing plants in the spring and reach grazable height before most of the grasses. As larkspurs are palatable to livestock, they are grazed readily and are very dangerous

during the early season, until the plants wither. All parts of the plants are poisonous. Although sheep graze the plants, they are seldom poisoned.

TOXIC PRINCIPLE Like tall larkspur, low larkspur contains several alkaloids. Symptoms and other details of poisoning are similar to those caused by tall larkspurs.

Timber milk-vetch (Astragalus miser Dougl. ex Hook. var. serotinus (Gray) Barneby) Fig. 4

DESCRIPTION Timber milk-vetch is a member of the pea family and has the characteristic pea flower and pod. It is a long-lived, attractive, fragile, taprooted plant that grows in tufts 20-50 cm tall. It is fine stemmed with compound pinnate leaves that carry 7-13 small (10-18 mm), slightly hairy oval-shaped leaflets. The pinkish mauve flower is generally 6-12 mm long, with few to many occurring along the flower stalk. Timber milk-vetch flowers from about mid-June, through July, and into August at higher elevations. The seed pod is 20-25 mm long. The small, black or greenish seeds mature between mid-July and mid-August and are shed soon after.

DISTRIBUTION AND HABITAT In British Columbia timber milk-vetch occurs in the fescue grasslands; in the western yellow pine, lodgepole pine, and Douglas-fir forests of the Cariboo and Chilcotin regions; and in the Thompson, Nicola, Okanagan, Similkameen, Kettle, and Columbia-Kootenay valleys. Although much less common than in British Columbia, it also occurs in the fescue grasslands of the foothills of the Rocky Mountains in southern Alberta.

CONDITIONS OF POISONING The toxicity of timber milk-vetch seems to differ from area to area, and some animals seem to be more susceptible than others. Some factors apparently affect toxicity significantly. For example, heavy rain after a dry period can increase toxicity twofold; the growth period just before flowering shows the highest toxicity and toxin levels decrease steadily thereafter; plants in fescue grassland appear to be more toxic than those in forested sites; plants in Douglas-fir forests show the lowest toxicity; and lactating cows may be more susceptible to poisoning than dry cows or male stock. Timber milk-vetch is eaten readily only when enough good forage is not available. However, once cattle have started grazing the plant, they appear to become addicted to it.

TOXIC PRINCIPLE Timber milk-vetch belongs to a group of milk-vetches that forms nitro compounds. In timber milk-vetch the toxic principle has been identified as miserotoxin, a glycoside which releases the highly poisonous 3-nitropropanol in the rumen of cattle and sheep.

SYMPTOMS AND LETHAL DOSE Early signs of poisoning include placidity and stupefaction. Animals tend to close the eyes partially and twitch the ears; excessive frothy salivation is evident. Instability and continual shifting of weight from one leg to the other, a slow and elongated gait, and high-headed carriage may also be observed.

When a large dose has been ingested over a short period, poisoning is acute. This form of poisoning is characterized by a progression of symptoms to include staggers, labored breathing, cyanotic mucous membranes, and usually sudden collapse. Grazing of timber milk-vetch over a prolonged period at low levels causes chronic poisoning, characterized by general depression, knuckling of the fetlocks, interference of the hindlimbs causing a clicking sound, arching of the back, partial paralysis, difficulty in respiration, protruding tongue, nasal discharge, and a roaring or wheezing sound.

Sudden exertion may cause death in both acute and chronic poisoning. In cattle, the lethal dose of 3-nitropropanol is about 3 g/100 kg body weight; less than 2 kg of timber milk-vetch may contain the lethal dose for a mature animal.

TREATMENT When poisoning is detected early and animals can be moved to areas where safe forage and fresh water can be provided, intramuscular injections of thiamine hydrochloride at 90 mg/100 kg body weight have been successful in some cases.

Spotted water-hemlock (Cicuta maculata L.; Synonym: C. douglasii (DC.).
Coulter & Rose, western water-hemlock) Fig. 5

DESCRIPTION Spotted water-hemlock is a rather pale green, branching, smooth perennial herb of the carrot family. The stems are stout, growing to 2 m tall, hollow, and jointed, and they often have purplish spots. The base of the stem is somewhat swollen and is characterized by hollow chambers divided by horizontal membranes. The leaves are twice compound and alternate; the leafstalk bears several leaflets in pairs on opposite sides. The leaflets are toothed on the margins. The flowers are small, white, and arranged in umbrella-like heads. The fruit is dill-like, small and round, with prominent coky ribs, and splits when mature. The plants flower in June and July, but seed does not ripen until late in the season. Plants usually have several rootstocks attached to the base of the stem; these are short and tuberous, exuding a yellowish, aromatic, extremely poisonous oil. Spotted water-hemlock is probably the most poisonous plant of the region.

DISTRIBUTION AND HABITAT Spotted water-hemlock occurs throughout Western Canada in slough margins, marshy areas, and wet meadows and along streambanks.

CONDITIONS OF POISONING Spotted water-hemlock is among the earliest plants to produce green foliage in spring, before other palatable forage is available. Because of its occurrence in wet habitats, grazing of the young foliage often results in the roots being pulled out of the soft soil. As both leaves and roots can contain a lethal dose of toxin, death can result in a few hours after ingestion of the plant. Hemlocks are poisonous to all livestock, as well as to human beings. Poisonings of humans are on record throughout countries in which Cicuta occurs. A small amount of the oily substance, licked from a root, can cause severe illness, or even death, in children.

TOXIC PRINCIPLE The toxic principle is contained in the oily substance

secreted from the roots as well as the young growth. It is a highly unsaturated higher alcohol and has long been known as cicutoxin. It is related to poisonous substances in other members of the carrot family.

SYMPTOMS AND LETHAL DOSE Symptoms of poisoning appear rapidly, usually within 30 min after ingestion of a lethal dose. The first symptom is excessive salivation and frothing at the mouth. Frothing is followed by tremors, uneasiness, and violent convulsions. Severe pain, especially in the abdomen, is evident; clamping of the jaws and grinding of the teeth often occur, and the tongue may be chewed and damaged.

The lethal dose is small: a single root can contain enough poison to kill a cow. Early spring growth is also extremely poisonous, but toxicity decreases with advancing growth.

TREATMENT Treatment is rarely possible. Morphine injections may be used to aid in controlling convulsions. A purgative may help in eliminating the poison.

Poison hemlock (Conium maculatum L.)

Fig. 10

DESCRIPTION Poison hemlock is another member of the carrot family and can be confused with spotted water-hemlock. However, the latter is a perennial, whereas poison hemlock is an annual or biennial. It grows to 3 m tall and has a smooth, stout stem, clearly purple spotted. Its leaves are large and much divided, resembling those of carrots; when crushed they exude a strong odor. The root of poison hemlock is large, white, and parsniplike. In contrast to spotted water-hemlock, the base of the stem and the root are not chambered.

DISTRIBUTION AND HABITAT Poison hemlock is not common in Western Canada. Although it requires ample moisture, it is not semiaquatic and has been grown in gardens for its fernlike leaves.

CONDITIONS OF POISONING Poison hemlock has been known as an extremely poisonous plant since ancient times; an extract from the plant was used to carry out death sentences in ancient Greece and Rome. In contrast to spotted water-hemlock in which the roots are the most poisonous organs and in which the leaves become less poisonous during the growing season, the leaves of poison hemlock accumulate more of the alkaloids than does the root and are most poisonous at maturity. The seeds of poison hemlock may have a very high concentration of poison. Poisoning of humans has occurred when the leaves of poison hemlock were mistaken for those of parsley, which they resemble.

TOXIC PRINCIPLE The toxic principle in poison hemlock consists of at least five alkaloids, including coniine.

SYMPTOMS AND LETHAL DOSE Symptoms usually appear within 1 h after ingestion of the poison. They progress from nervousness and trembling, to loss of control of the limbs and dilation of the pupils, to slowing of the heartbeat, coldness, and coma and finally to death through respiratory failure. Poison hemlock is



Fig. 10 Poison hemlock



Fig. 11 Upper, water parsnip; lower, angelica sp.

extremely poisonous, and fresh plant material equivalent to about 0.25% of an animal's liveweight can constitute a lethal dose.

TREATMENT Stimulating the heartbeat, if done early enough, may prevent death in some cases.

RELATED SPECIES Spotted water-hemlock and poison hemlock may also be confused with water-parsnip, with which it often grows in slough or lake margins. The plants can readily be distinguished, however, by characteristics of leaves, stems, and roots. Water-parsnip (Fig. 11, upper), Sium suave Walt., has once-divided leaves; the leaflets are long, almost linear, and sessile. The stem is not enlarged at the base and not conspicuously chambered in cross-section. In spotted water-hemlock the leaflets are stalked and divided in three to five segments, and the enlarged stem base is conspicuously chambered. The inflorescences of two other members of the carrot family resemble those of spotted water-hemlock and poison hemlock. Cow-parsnip, Heracleum lanatum Michx., grows in shrubbery and moist areas; it is not semiaquatic, as is spotted water-hemlock. Its leaves are usually very large and divided into three large leaflets, and the entire plant is hairy. Angelica (Fig. 11, lower), Angelica spp., occur in moist meadows and forests but are not common in areas where hemlocks are found. Their leaves are twice divided, with the divisions having three to seven leaflets.

Nitrate poisoning

Several common plants, including some forage plants, can cause livestock poisoning through accumulation of large amounts of nitrates. The most common cause of nitrate poisoning is oat hay or straw, but barley and wheat straw, as well as corn, flax, sorghum, and other forages, have been found to contain toxic quantities of nitrate. Several weedy species, including lamb's-quarters, kochia, lady's-thumb, and smartweeds, can also contain large amounts of nitrate.

CONDITIONS OF POISONING Nitrate accumulation in plants can have several different causes. Drought late in the growing season can result in the accumulation of nitrates in immature oats; high levels of fertilization with nitrogen may result in high nitrate levels in grasses. Forage crops or weedy species growing on areas with high levels of nitrate such as feedlots, cattle yards, and dried-up sloughs may be dangerous. Hay from such areas, particularly from those which are shaded, may also be hazardous, as nitrate accumulation increases with increased shading. Animals usually prefer feed high in nitrates and are likely to ingest large amounts of this feed. Animals on poor-quality feed are more susceptible to nitrate poisoning than those on good-quality feed. Susceptibility to poisoning can be reduced by the supplementing of carbohydrate concentrates, taking care that animals cannot select only the high-nitrate feed.

TOXIC PRINCIPLE When plants with a toxic amount of nitrate are consumed by livestock, a conversion of nitrate to nitrite takes place in the intestinal tract. This nitrite is absorbed into the bloodstream, where it combines with

the hemoglobin, the oxygen carrier of the blood, to form methemoglobin. Because methemoglobin does not combine with oxygen, the oxygen-carrying capacity of the blood is reduced to the extent that affected animals may die of anoxia. Because of the rapid course of the illness, nitrate poisoning may be confused with anthrax, clostridial diseases such as blackleg or malignant edema, or pasteurella infection such as hemorrhagic septicemia.

SYMPTOMS AND LETHAL DOSE Typical symptoms of nitrate poisoning in cattle include restlessness, frequent passing of urine, and extreme weakness. The blood becomes dark brown. Finally the animals collapse, sink to the ground, roll on the side, and die quietly without a struggle. A post mortem shows hemorrhages and inflamed areas on the surface of the rumen and small intestines. Forages with over 0.34-0.45% nitrogen in the nitrate form are potentially toxic.

TREATMENT Intravenous injection of a 1-4% aqueous solution of methylene blue should be given at the rate of 1 g/100 kg of animal body weight. Because of its irritant properties, methylene blue should not be allowed to escape into the tissues surrounding the vein.

Photosensitization

Several plant species may cause photosensitivity in animals when eaten in large amounts. The animals become sensitive to sunlight owing to the action of pigments in the peripheral circulation. The pigments may be derived from the plants or are produced through liver dysfunction. However, the symptoms are identical, or nearly so, in all cases. A sensitized animal reacts to sunlight with reddening of the skin in unpigmented areas. The affected areas become itchy, followed by the swelling and eventually dying off of the skin tissue. White cattle are most affected, and susceptibility decreases with increasing pigmentation; black animals are usually not affected. Recovery is usually rapid after changing feed and keeping animals protected from direct sunlight.

The three plants that are most commonly linked with causing photosensitization in animals are described below.

St. John's-wort, Hypericum perforatum L., also known as goatweed, is an unpalatable perennial, which grows 20-50 cm tall. It has small leaves pitted by small glands in the undersurface. The yellow flowers occur in clusters and develop into many-seeded brown capsules. The plants spread by strong underground rootstocks and often invade even lightly grazed grassland ranges. The species occurs in British Columbia in several areas but has not been found in the Prairie Provinces.

Lady's-thumb, Polygonum spp., has oval or lanceolate leaves and pale pink flowers in erect spikes. There is a membranous sheath at the junction of leaf and stem. The plant is locally common in shallow water or near the margins of sloughs and meadows. It is usually associated with a mixture of grasses and sedges. If eaten when green, it can cause photosensitization in animals. Water smartweed, Polygonum amphibium L., also can cause photosensitivity; it

occurs in sloughs, marshy areas, and shallow water.

Horsebrush, Tetradymia canescens DC., is a low, spreading, pale green to gray shrub with soft, needlelike leaves and deep yellow flowers in clusters. It closely resembles and generally grows with rabbitbrush. Known only in British Columbia, it seldom occurs in sufficient abundance to cause trouble. The plant is occasionally found on moderately dry grasslands on lighter soils.

Other plant species that have caused photosensitivity include turnip greens, oats, panic grasses, sudan grass, alfalfa, vetches, and kochia. Drinking water showing a bloom of blue green algae also can sensitize animals.

Selenium poisoning

Some plants growing on soils containing the element selenium accumulate this element in considerable amounts. These plants become potentially toxic when they contain selenium at levels of 5 ppm or more and are ingested over a prolonged period. Acute poisoning, caused by the ingestion of large amounts of plant material with a very high selenium content (up to 15 000 ppm) is rare because animals do not eat these plants unless forced by starvation. Hence, acute selenium poisoning occurs primarily on severely overgrazed pastures on seleniferous soil. Chronic poisoning can be of two types, known as blind staggers and alkali disease. The primary cause of both forms is the ingestion of milk-vetches, but some of the associated plants can also accumulate low levels of selenium.

Blind staggers occurs when animals consume selenium-containing plants over a considerable period. Animals lose muscular coordination, have impaired vision and paralysis, and move about restlessly, bumping into fences or corrals.

In alkali disease, animals lose their appetite and get thin. Their hooves become deformed and may drop off. There is a general loss of hair, particularly about the tail, and sores develop. Reproduction is impaired and animals may be sterile.

The two species of milk-vetch that are linked with selenium poisoning are described below.

Narrow-leaved milk-vetch (Fig. 12, upper left), Astragalus pectinatus (Hook.) Dougl., is an erect or semierect, much-branched herb that grows 30-50 cm tall. The leaves are compound pinnate with 11-21 very narrow leaflets 12-50 mm long. The flowers open in early June. There are 5-20 very noticeable cream-colored flowers 20-25 mm long on each of several racemes. The seed pods are woody, oblong, ellipsoidal, and 10-15 mm long. This plant is found in southwestern Manitoba, southern Saskatchewan, and southern Alberta on open prairies and roadsides, usually on lighter soils.

Two-grooved milk-vetch (Fig. 12, upper right), Astragalus bisulcatus (Hook.) A. Gray, is a stout, many-stemmed, erect plant 30-80 cm high. It has



Fig. 12 Upper left, narrow-leaved milk-vetch; upper right, two-grooved milk-vetch; lower, silky lupine.

a distinct, unpleasant odor. The leaves have 17-27 elliptic leaflets 10-20 mm long. The flowers are showy, deep purple, and about 10 mm long and grow in long, dense racemes at the ends of long stems. The seed pods are 10-15 mm long and have two deep grooves along one side. This plant is very common throughout southern Saskatchewan and Alberta. It is usually found on semimoist sites. It may be abundant in ditches along new roads. Heaviest stands occur on shallow soils covering shale. To treat affected animals, remove them from the area or provide supplemental feed known to be free from or low in selenium.

SECONDARY TROUBLEMAKERS

Early yellow locoweed (Oxytropis sericea Nutt. var. spicata (Hook.) Barneby) Fig. 6

DESCRIPTION This species is a member of the pea family. Early yellow locoweed is low growing. The leaves are composed of 7-21 oval leaflets 12-20 mm long, which have silky or short hairs. The yellow flowers are 15-20 mm long. Lower conjoined petals have a characteristic sharp erect point at the tip. The seed pods are about 20 mm long and are covered with short hairs that are mostly white but a few black. It is a showy plant, very noticeable in early spring. There are other locoweeds with yellow, cream, or purple flowers; all have the sharp erect point on the lower petals.

DISTRIBUTION AND HABITAT Early yellow locoweed and several other poisonous locoweeds are found throughout Western Canada and in various habitats. Suspected plants should be taken or sent to the nearest Agriculture Canada research station for identification.

CONDITIONS OF POISONING Locoism, caused by this plant, is uncommon today, but it was fairly common in early settlements. Locoweeds are seldom eaten unless range is severely overgrazed. Although locoweeds are unpalatable when sufficient other forage is available, animals forced to eat it become addicted to the plants and actually seek them out.

TOXIC PRINCIPLE Despite numerous studies, the toxic principle of locoweeds is still unknown. Recent studies indicate, however, that indolizidine alkaloids are the cause of locoism.

SYMPTOMS AND LETHAL DOSE Animals become listless and passive, and they exhibit irregularities in gait and eating. In the later stages of the disease, animals stop eating and eventually they die. Symptoms appear in cattle and sheep after ingestion of about 90% of the animals' weight, and death results after ingestion of more than three times the body weight over a period of about 3 months. Horses are more susceptible, showing symptoms after ingestion of about 30% of the body weight over a period of about 6 weeks.

TREATMENT Remove animals from infested areas and give them palatable and nutritious feed until they recover.

Choke cherries (Prunus virginiana L., incl. var. melanocarpa (Nels.)
Sarg.) Fig. 7

DESCRIPTION Choke cherries are tall, deciduous shrubs of the rose family, growing to 2-6 m. The small, white flowers grow thickly in terminal clusters. The leaves are smooth and simple, with a toothed edge. The small fruits are red or purplish; they have a bitter, astringent taste and a stonelike seed. Flowering generally occurs in May and June; the fruit remains on the shrub until late fall.

DISTRIBUTION AND HABITAT Choke cherries occur throughout Western Canada. They are common in moist coulees and on creek banks and occur abundantly on light soils.

CONDITIONS OF POISONING Choke cherries are somewhat unpalatable to livestock, but they are browsed when other forage is scarce or when the new foliage emerges in the spring. The toxicity of the plant varies considerably, but the foliage is toxic at all stages of growth, and the buds, flowers, and twigs are potentially dangerous.

TOXIC PRINCIPLE Choke cherries produce the cyanogenic glycoside prunasin, which when ingested releases prussic acid through the action of enzymes in the rumen.

SYMPTOMS AND LETHAL DOSE Uneasiness, shivering, staggering, and difficulty in breathing are the first symptoms. These may occur within an hour of browsing on choke cherry. In severe cases, the heartbeat becomes very rapid, mucous membranes show a distinct pink color, and convulsions develop, followed by death. Because of the extreme toxicity of HCN the lethal dose is low, about 0.25% of an animal's body weight of fresh or young foliage.

TREATMENT Animals usually die so soon after browsing on choke cherry that treatment cannot be given. When poisoning is detected very early, sodium nitrite and sodium thiosulfate therapy should be tried.

Saskatoon (Amelanchier alnifolia Nutt.)

Fig. 8

DESCRIPTION Saskatoon is a shrub or small tree growing 1-4 m high, in the rose family. The leaves are simple, toothed only at the tip to about halfway along the margin. The flowers are white and grow in rather open, few-flowered racemes. The fruit is a sweet, purplish berry.

DISTRIBUTION AND HABITAT Saskatoon occurs throughout Western Canada in moist coulees, along creeks, and in open woodlands. It often occurs together with choke cherry.

CONDITIONS OF POISONING Saskatoon begins growth early in the spring, commonly before much grass is available for grazing, and somewhat earlier than choke cherry. The buds and young foliage are highly toxic, especially during the early flowering stage. Dormant twigs can also be toxic.

The toxic principle and poisoning symptoms are like those of choke cherry.

Silky lupine (Lupinus sericeus Pursh)

Fig. 12

DESCRIPTION Silky lupine is a beautiful, rather stout, taprooted perennial forb. It grows as high as 60 cm and is somewhat bushy in appearance. The foliage is silvery green because of the dense covering of short, fine hairs on the stems and leaves. Five to eight leaflets, which arise from the top of the leaf stem, present a palmlike appearance, as they are fairly long (up to 5 cm) and narrow. The leaf stems are alternate on the stalk. The many flowers are blue, about 6-15 mm across, and distinctly pealike. They have short stalks and are arranged in dense clusters along the spikelike stem. The fruit pod is about 3 cm long and densely covered with short, silky hairs. The taproot is fairly long and forms a crown, as in alfalfa. The plant blooms in June and July and the seeds mature in July and August. The plant remains green throughout the growing season.

DISTRIBUTION AND HABITAT Silky lupine occurs in fescue prairie in southwestern Alberta, and in the fescue grasslands and yellow pine zones in the interior of British Columbia. It is found on open grassy hillsides, generally in rich, light, well drained soils. It is common to abundant and is scattered over large areas, which makes local control difficult.

CONDITIONS OF POISONING The seeds and pods of lupines are the most poisonous parts, but the leaves may cause trouble when large quantities are eaten at one time. Most poisonings occur when hungry stock, usually sheep, are turned onto infested areas.

TOXIC PRINCIPLE The poisonous principle consists of alkaloids of the quinolizidine group. The poison is not cumulative and is readily excreted when less than a lethal dose is ingested in one feeding. The quantity of alkaloids in plants varies with soil and climate conditions. The alkaloids remain present after drying, and hay containing large amounts of lupines may be highly dangerous when pods with seeds are present. Another alkaloid may be present, which causes crooked calf disease in pregnant cows grazing lupines between the 40th and 70th day of gestation.

SYMPTOMS AND LETHAL DOSE Poisoning causes heavy, labored breathing, which is followed by coma or frenzy, frothing at the mouth, and violent spasms. When poisoned by lupine, sheep butt anything in their way. Sheep can safely eat up to 10 kg of the leaves but a dose of 750 g of the seed pods and seeds is likely to cause death.

TREATMENT No specific remedy is known. When used early, potassium permanganate may be useful as a chemical antidote. Morphine and other sedatives are useful.

RELATED SPECIES Silvery lupine, Lupinus argenteus Pursh., is similar to silky lupine, but as the hairs on the stems and leaves lie flat the plants

appear less woolly. Flowers are light violet or purplish to almost white.

Silvery lupine is plentiful in Cypress Hills-Wood Mountain and the foothills of the Rocky Mountains and is less common throughout southern Alberta along the Milk River Ridge.

Its poisonous characteristics and the treatment are the same as for silky lupine.

Greasewood (Sarcobatus vermiculatus (Hook.) Torr.)

Fig. 13, upper left

DESCRIPTION A much-branched shrubby perennial with spiny branches, greasewood may grow to 1 m but a height of 50-80 cm is more common. The leaves are pale yellowish green and fleshy, about 25-35 mm long. The very small male and female flowers are borne separately. The male flowers are in small spikes at the ends of the stems; the female flowers are borne singly in the axils of the leaves. The seed is surrounded by a broad membranous wing.

DISTRIBUTION AND HABITAT Greasewood is found only in the southern part of Alberta and Saskatchewan on strongly saline flats.

CONDITIONS OF POISONING Lambs are the animals most subject to greasewood poisoning. The losses occur soon after flocks are moved to summer range, when the animals are hungry and are in unfamiliar surroundings. Sheep and calves have also been poisoned by greasewood.

TOXIC PRINCIPLE The buds and young leaves of greasewood contain salts of oxalic acid.

SYMPTOMS AND LETHAL DOSE Intake of greasewood causes depression, weakness, shallow pulse and breathing, and collapse. The lethal dose is estimated at 1.5-5% of an animal's weight, depending on hunger and general condition of the animal.

Horsetails (Equisetum spp.)

Fig. 13, upper right

DESCRIPTION Horsetails are well-known, nonflowering perennial herbs, also known as scouring-rushes. The aerial stems are 20-60 cm tall, rushlike, ridged, jointed, and generally hollow and single, ending in a cone or with whorls of four-angled, fine, green branches. They contain a rather large amount of silica, which makes them very harsh. The leaves are very small and scalelike, and they form a cylindrical sheath at the nodes or joints of the stems.

DISTRIBUTION AND HABITAT Horsetails are common in moist fields, swales, and meadows and around lakes and sloughs throughout Western Canada, especially in flood plains and light soils. They are a common component of native meadow and slough hays.

CONDITIONS OF POISONING Horses, mainly, are affected, especially by eating hay



Fig. 13 Upper left, greasewood; upper right, horsetail sp.; lower left, bracken; lower right, monk's-hood.

that contains much horsetail. Trouble in pastures is rare, but cattle and sheep can be affected by grazing large amounts, as well as by hay containing a large proportion of horsetail.

TOXIC PRINCIPLE In horses, poisoning appears to be caused by thiaminase, an enzyme that destroys thiamine. However, thiaminase cannot account for poisoning in ruminants, and the toxic principle affecting cattle and sheep is not known.

SYMPTOMS AND LETHAL DOSE Symptoms of the poisoning are unthriftiness, loss of weight, and gradual weakening of the animal. After an animal has fed on horsetails for 2-5 weeks, it loses muscular control, falls down, and struggles violently to get up. The animal is usually willing to eat but is unable to rise and finally dies of exhaustion. Grain-fed animals are able to resist the action of the poison better than animals not fed grain. The symptoms may be slow to develop, as the speed of development depends upon the age of the animal and the amount of horsetail in the hay.

TREATMENT Immediately stop feeding hay that contains horsetail. Give a purgative and bran mashes to assist in removing the poisonous plant from the digestive tract. Large doses of thiamine are effective in horses so long as animals are still able to get up.

Horsetails occur most commonly in poorly drained fields. To eliminate or reduce the hazard, provide good drainage, then cultivate and reseed the fields.

Bracken (Pteridium aquilinum (L.) Kuhn)

Fig. 13, lower left

DESCRIPTION Bracken is a tall, coarse perennial fern, growing 50-125 cm. The aboveground parts of the plant, the fronds, have the appearance of large, finely divided, broadly triangular leaves with a strong, somewhat woody central stem. The fronds develop by unrolling. The underside of the fronds is covered with fairly coarse, brown hairs. The margins of the fronds are rolled under and lined with tiny, rust-colored spore cases. The plant reproduces by means of stout, black, woody rootstocks and from spores.

DISTRIBUTION AND HABITAT Bracken is found only in eastern Manitoba, the Riding Mountains, southwestern Alberta, and British Columbia. In British Columbia it is generally associated with western red cedar and western hemlock forests. It is locally common to abundant in upland pastures, abandoned fields, disturbed areas, and forested and burned-over areas in regions with high rainfall, especially on poorly drained gravelly or sandy soils.

CONDITIONS OF POISONING Bracken is poisonous when in green forage and dried forms, so it may be a problem in hay. It is not palatable and usually is not eaten unless other forage is scarce. Cattle and especially horses are often affected by eating hay that contains a large proportion of bracken.

TOXIC PRINCIPLE Bracken contains several potential toxins, including thiaminase, an enzyme that is also present in horsetails. Also present are

factors causing aplastic anemia.

SYMPTOMS AND LETHAL DOSE Horses fed on bracken-containing hay become lethargic, lose weight, and show incoordinated movement. In the later stages tremors develop and the animals are unable to remain standing and unable to get up despite frantic efforts. The lethal dose is large, about 80% of an animal's weight ingested over 1 month or less. In ruminants symptoms of poisoning of mature animals differ from those in calves. Initially, animals lose condition, and may have excessive mucous discharge from the mouth and nose. In mature animals these symptoms are followed by depression, loss of appetite, and inflammation of the intestines with frequent blood clots in the feces. In later stages there may be bleeding from the nostrils and elevated body temperature shortly before death. In calves, the initial symptoms are followed by difficulty in breathing with characteristic roaring, as well as elevated temperature. The lethal dose may be as high as the body weight of an animal, ingested over a prolonged period. In ruminants, symptoms of poisoning may not appear until several weeks after removal from feed containing bracken.

TREATMENT Horses can be treated with daily doses of 100 mg of thiamine, injected intramuscularly; this treatment is especially effective when begun in early stages of poisoning. No effective treatment for cattle exists. Recovery of both horses and cattle is aided by removing them from feed containing bracken and providing them with good-quality feed.

Monk's-hood (Aconitum columbianum Nutt.)

Fig. 13, lower right

DESCRIPTION This plant belongs to the buttercup family and is closely related to the larkspur, which it resembles. It looks much like tall larkspur, but it can be distinguished by the hood or helmet formed by one petal over the rest of the flower in place of the spur. Roots of monk's-hood are short and tuberlike with yellowish rootlets, whereas those of tall larkspur are long and fibrous. The stem of monk's-hood is pithy, whereas the stem of larkspur is hollow. The upper leaves of monk's-hood are close to the stem; those of larkspur have petioles or stalks.

DISTRIBUTION AND HABITAT The plant has much the same distribution and habitat as that of tall larkspur, and often both are found growing together, though monk's-hood is much less common. Monk's-hood occurs in mountain meadows in the Rocky Mountains of Alberta and British Columbia. Some species are also used as ornamental plants in gardens.

CONDITIONS OF POISONING The plant appears to be most poisonous before flowering but can cause poisoning until fall. The young, emerging plants are especially dangerous.

TOXIC PRINCIPLE Toxicity is due to the presence of several diterpenoid alkaloids, including aconitine. The roots and seed are especially high in toxins.

SYMPTOMS AND LETHAL DOSE Symptoms of poisoning are muscular weakness,



Fig. 14 Heaths: upper left, western minniebush; upper right, Labrador-tea; lower left, white rose-bay; lower right, bog-laurel.

irregular and labored breathing, weak pulse, bloating and belching, constant attempts at swallowing, and contraction or dilation of the pupils of the eyes. The lethal dose depends mainly on the plant parts ingested. Less than 1% of an animal's body weight in fresh roots or seeds can cause death.

TREATMENT No antidote is known. Drugs such as digitalin or atropine are given to stimulate breathing and to overcome depression of heart activity.

Heaths (Ericaceae)

Fig. 14

Several shrubs of the heath family are poisonous to livestock. They are grouped together because they have somewhat similar growth habits, distributions, toxic principles, and symptoms of poisoning.

western minniebush	<u>Menziesia ferruginea</u> Sm.
white rose-bay	<u>Rhododendron albiflorum</u> Hook.
trapper's-tea	<u>Ledum glandulosum</u> Nutt.
Labrador-tea	<u>Ledum palustre</u> L.
bog-rosemary	<u>Andromeda polifolia</u> L.
bog-laurel	<u>Kalmia polifolia</u> Wang.

DESCRIPTIONS Western minniebush is a medium-sized, branching shrub 1-2 m high, with thin, alternate leaves that have scattered, rusty hairs on the upper surface. The flowers are greenish purple and rather small, and are borne in terminal clusters.

White rose-bay is also a medium-sized shrub with thin, clustered leaves. The flowers, one to three in a cluster, are showy, pale yellow, and bell-shaped, about 3 cm across.

Trapper's-tea is a low shrub with fairly thick leaves that are resin-dotted underneath; the resin makes the herbage fragrant when bruised. The flowers are small and yellowish white, and are borne in terminal clusters.

Labrador-tea is very much like trapper's-tea, except that its leaves are densely hairy below instead of glandular.

Bog-rosemary grows to about 50 cm tall. The leaves are small, linear-oblong, and white felty-hairy below, with the margins rolled in; the leaves are alternate on the stem.

Bog-laurel is a somewhat smaller, branching, evergreen shrub 25-60 cm high, with oblong, leathery, opposite leaves that are dark glossy green above and whitish beneath. The leaf margins are rolled under. The flowers are small and lilac-colored, and are borne in terminal clusters.

DISTRIBUTION Western minniebush and trapper's-tea occur in the southern Rocky Mountains and in British Columbia, mostly in wet meadows and bogs. White rose-bay occurs in the same areas, but in drier situations. Labrador-tea, bog-rosemary, and bog-laurel are found in muskeg areas and damp forest throughout Western Canada.

CONDITIONS OF POISONING Sheep are the most commonly affected livestock. The plants are seldom grazed by cattle unless other feed is short, and most of the localities where these plants are found are used as sheep range. Because the leaves of most of these species are leathery or bitter, their palatability is rather low.

TOXIC PRINCIPLE Ericaceous shrubs produce grayanotoxins, which are poisonous diterpenoids.

SYMPTOMS AND LETHAL DOSE Symptoms of poisoning are salivation, general depression, increased flow of secretions from the nose, convulsions, paralysis of the limbs, and failure of the central nervous system.

TREATMENT Animals should be removed from infested areas and given laxatives, demulcents, and nerve stimulants.

Mushrooms

Livestock may be poisoned by certain mushrooms, although mushroom poisoning is uncommon in Western Canada. However, when mushrooms are abundant, stock may develop a taste for them and eat considerable quantities. These animals then become emaciated from poor nutrition rather than from poison.

Stock poisoning by mushrooms is most likely to occur in wooded range, where the fly agaric, Amanita muscaria (L.) Fr., occurs and may be ingested when growing among grasses. Only a few fresh mushrooms constitute a lethal dose, and death follows severe pain and convulsions.

Algae

Every slough or dugout, except those of extreme salinity, contain algae in greater or lesser amounts. Most of these do not pose any problems, but under certain conditions a few species can cause trouble, especially members of the blue green algae. These species exude toxic substances into the water, and drinking of the water can cause severe illness or death. Also, because blue green algae are able to fix nitrogen, nitrate poisoning or photosensitization can result from drinking water with large populations of these algae. Very rapid growth of algal populations can occur when drinking water becomes polluted with nitrogen and phosphorus-containing fertilizers. Under pasture conditions, the excrement of drinking animals can be the source of this pollution, especially when precipitation is low and water levels are well below normal. The presence of algae is evident in pond scum or as large clumps floating below the surface.

No treatment for poisoned animals is available. However, the growth of algae can be prevented by treating the water with chlorine or copper sulfate (bluestone) at a rate of 1 ppm.

Western yellow pine (Pinus ponderosa Dougl.)

DESCRIPTION Western yellow, or ponderosa, pine is a coniferous tree. Under average range conditions it grows 20-25 m high. The dark green needles are 15-30 cm long and they occur in bundles of two to five needles, usually three. The tree has a characteristic reddish orange platy bark.

DISTRIBUTION AND HABITAT It is a chief component of the vegetation of the subhumid region of southern interior British Columbia.

CONDITIONS OF POISONING Under certain conditions fresh or dry needles of western yellow pine can induce abortion in cattle, usually in the last 3 months of pregnancy. The problem areas are confined to the yellow pine belt of the interior valleys of the Fraser, Nicola, Thompson, Similkameen, and Okanagan rivers, eastward along the international boundary to Nelson and the southern portion of the Rocky Mountain Trench.

TOXIC PRINCIPLE The substances causing abortion in cattle are not known. Studies with mice indicate that the agents causing abortion may be microorganisms, mycotoxins, embryotoxins, or antiestrogens.

SYMPTOMS AND LETHAL DOSE Premature births, stillbirths, weak calves that die shortly after birth, and retained placenta can occur as a result of poisoning by yellow pine. Maternal toxicity and death have also been reported.

TREATMENT The bred cow herd should be wintered in areas free from yellow pine. When this is impossible, prune the lower branches of the trees and clear the pasture of fallen trees after storms. Discontinue logging operations in pastures where bred cows are grazed because cows nibble on the slash even when they are adequately fed.

MECHANICALLY INJURIOUS PLANTS

Some common plants, although not poisonous, occasionally cause considerable injury to livestock. They have sharp seeds, awns, or spines that may work their way into the tongue, gums, eyes, nose, or skin. This injury may result in sores that cause extreme discomfort or inflammation. Suffering animals go off feed, which causes them to lose weight and to develop a generally poor condition. The sores may also allow the entry of pathogenic bacteria into the tissues and circulatory system, causing local or general infections. The fibers of some plants, e.g. foxtail barley, may form balls that lodge in the stomach or intestines. The following plants present a risk of mechanical injury for livestock.

downy brome	<u>Bromus tectorum</u> L.
needle-and-thread	<u>Stipa comata</u> Trin. & Rupr.
northern porcupine grass	<u>Stipa curtiseta</u> (Hitchc.) Barkworth
porcupine grass	<u>Stipa spartea</u> Trin.
red three-awn	<u>Aristida longiseta</u> Steud.
foxtail barley	<u>Hordeum jubatum</u> L.
wild oats	<u>Avena fatua</u> L.

common burdock
prickly-pear

Arctium minus (Hill) Bernh.
Opuntia spp.

POSSIBLE TROUBLEMAKERS

The following plant species, which occur commonly within the ranching areas of the Western Provinces, are reported to have caused poisoning among stock in other regions, although no losses from these plants have been brought to our attention in the prairies. Ranchers should watch these species carefully and report any losses so that they can be investigated by local authorities.

Black nightshade, Solanum nigrum L., is a much-branched, somewhat spreading annual that has oval-shaped leaves with pointed tips and wavy margins. The clustered, whitish flowers resemble those of a potato. The berries are round, smooth, and green; they turn black when mature. The plant is a weed that grows in moist fields and waste places, including areas around corrals and barnyards, especially on lighter soils. It does not normally grow on areas with a perennial plant cover. The toxic principle consists of glycoalkaloids, and α -solanine is the principle toxin. Although the lethal dose is small, the plant is rarely abundant enough to pose a problem.

Dogbanes, Apocynum spp., are perennial, creeping-rooted, erect, branching forbs. They have broad, opposite leaves and clusters of small, pink, bell-shaped flowers that produce long, slender pods containing numerous seeds with tufts of silky hairs. The stems and leaves contain a milky juice. Dogbanes are commonly found on abandoned fields, waste places, dry meadows, and open wooded areas. They generally occur on sandy or gravelly soils, especially in pine woods. Dogbanes contain several toxic cardiac glycosides, including cymarin. The plants are unpalatable, and ingestion is unlikely except when animals are starving.

Golden corydalis, Corydalis aurea Willd., has golden yellow flowers and smooth, often silver-tinged, deeply cut leaves. It closely resembles bleedingheart, Dicentra. It occurs commonly in moist, open woods but seldom in concentrations sufficient to cause trouble. The toxic principle consists of several alkaloids. Symptoms of poisoning are restlessness and twitching of the facial muscles, followed later by convulsive spasms. Typically, animals make biting or snapping movements throughout the period of poisoning. Sheep, especially, are susceptible.

Showy milkweed, Asclepias speciosa Torr., is an erect, showy, perennial herb that has strong, creeping rootstocks. The leaves are generally wide, somewhat leathery, and woolly, and they contain a milky juice. The flowers are pinkish mauve, clustered in round heads at the stem tips. The fruits are large, with thick coats enclosing numerous reddish brown seeds, each with tufts of long, silky hairs attached. The plants may be locally common in moist locations, pastures, abandoned fields, and similar areas throughout Western Canada. The toxic principle consists of cardiac glycosides. Milkweeds are eaten only when livestock is forced by starvation. Weakness, staggering, and

deep depression are symptoms of poisoning by milkweed, which appear within a few hours after ingestion. Violent seizures occur repeatedly after an animal falls down, and death follows within 24-48 h.

Tall buttercup, Ranunculus acris L., is a perennial, hairy buttercup growing to 80 cm tall, with round, divided leaves. It is common in pastures and meadows, along streams, and in waste places where sufficient moisture is available. It contains ranunculine, a glycoside present in several other species of Ranunculus, from which protoanemonin is released enzymatically. The amount of ranunculine in plants depends on the growth stage, but the toxin is usually highest at flowering when the concentration may be 1.5-2.5% on a dry weight basis. Fatal poisoning is rare, but animals eating buttercups may suffer irritation of the oral tissues and become either depressed or excited. Excessive salivation, diarrhea, and abdominal pain may be evident.

CONVERSION FACTORS

Metric units	Approximate conversion factors	Results in:
LINEAR		
millimetre (mm)	× 0.04	inch
centimetre (cm)	× 0.39	inch
metre (m)	× 3.28	feet
kilometre (km)	× 0.62	mile
AREA		
square centimetre (cm^2)	× 0.15	square inch
square metre (m^2)	× 1.2	square yards
square kilometre (km^2)	× 0.39	square mile
hectare (ha)	× 2.5	acres
VOLUME		
cubic centimetre (cm^3)	× 0.06	cubic inch
cubic metre (m^3)	× 35.31	cubic feet
cubic metre (m^3)	× 1.31	cubic yards
CAPACITY		
litre (L)	× 0.035	cubic foot
hectolitre (hL)	× 22	gallons
hectolitre (hL)	× 2.5	bushels
WEIGHT		
gram (g)	× 0.04	oz avdp
kilogram (kg)	× 2.2	lb avdp
tonne (t)	× 1.1	short tons
AGRICULTURAL		
litres per hectare (L/ha)	× 0.089	gallons per acre
litres per hectare (L/ha)	× 0.357	quarts per acre
litres per hectare (L/ha)	× 0.71	pints per acre
millilitres per hectare (mL/ha)	× 0.014	fl. oz per acre
tonnes per hectare (t/ha)	× 0.45	tons per acre
kilograms per hectare (kg/ha)	× 0.89	lb per acre
grams per hectare (g/ha)	× 0.014	oz avdp per acre
plants per hectare (plants/ha)	× 0.405	plants per acre

